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ELECTROCARDIOGRAPHIC FINDINGS IN THE PRESENCE OF MYOCARDIAL INJURY

The commonly-held view that displacements of the RS-T segment seen in many types of myocardial injury are directly the result of the current of injury is not true. Injury to heart muscle or other excitable tissue not only may wipe out more or less completely resting membrane potentials but also may prevent or modify excitation without causing other change.

If the injury is sufficient to make resting membrane potentials subnormal, the injured region will be negative with respect to normal resting muscle and a current, the current of injury, will flow between them. This current will flow chiefly in electrical diastole since, with excitation, normal muscle becomes depolarized and the potential difference between normal and injured muscle, responsible for the current of injury, disappears. If an electrocardiogram is taken with one electrode on the injured area and the other on normal muscle and, if the difference in potential between the two in diastole is neutralized by an equal and opposite voltage from the control box, this neutralizing voltage will produce displacement of the RS-T segment since the injury current disappears during electrical systole. The direction of this displacement will be opposite to that which the unopposed injury potential would produce.

If injury to the heart muscle prevents or modifies activation without materially altering the resting membrane potential, it is clear that during electrical systole the region affected will be positive with respect to normal muscle and upward displacement of the RS-T segment will be found in a tracing secured with the exploring electrode on or close to this area. No injury current, as this term is ordinarily used, will exist under these circumstances.

Curtis and Cole[1] by ingenious experiments performed on the giant axon of the squid have shown that, while alterations of the potassium content in the medium surrounding the axon affect both resting potentials and excitation, phenomena related to excitation are much more easily influenced by changes in the concentration of potassium than are resting membrane potentials. Their studies suggest that activation is not "a passive depolarization of the membrane or abolition of the resting potential" and they feel that a separate mechanism may be responsible for the production of each. During the passage of the activation wave along the axon the membrane potential was momentarily reversed in sign with the outside as much as 110 milli-volts negative with respect to the inside. These studies when extended and confirmed on other excitable tissues may greatly advance our knowledge in this basic field.

Bayley,[2] after consideration of the pathologic findings following myocardinal infarction and the electrical requirements pertaining thereto, has developed an attractive theory to explain observed deviations of the RS-T segment and the usual subsequent T wave changes. He clearly distinguishes between injured and ischemic muscle on the basis of the electrical effects produced by each. Injured muscle acts during diastole and systole "as the source of an electrical field of injury, and is a zone within which there exists or appears to exist a gradient in the intensity of polarization. The injured zone is responsible for the RS-T junction displacements which appear on the completed record only during systole. An ischemic zone is one in which polarization is everywhere complete during diastole (as in the case of normal muscle) but (unlike normal muscle) is one in which the onset of polarization is abnormally tardy during the regression period. The existence of an ischemic zone of sufficient dimensions accounts for the primary T wave changes which are occasionally observed before, and frequently after, myocardial infarction."

Bayley points out that soon after infarction occurs the zone of injured muscle lying between dead and ischemic muscle is relatively large but, with the passage of time, it usually shrinks in size as the ischemic region expands and soon vanishes leaving only the ischemic muscles between dead and healthy muscle. The ischemic zone usually decreases gradually in size and disappears weeks or months after infarct occurred. That ischemia may occur without injury and may precede, as well as follow the injury that results from coronary occlusion or narrowing helps to explain on a rational basis RS-T segment and T wave changes seen in coronary artery disease and other conditions. Clinical and experimental proof of these concepts has been provided by Bayley and Monte[3] and by Bayley, LaDue and York.[4]

An important part of Bayley's work is the suggestion that when infarction occurs the infarct may be relatively small and the areas of injury and ischemia large. It must be remembered that the infarct itself

is made up of dead or dying muscle all of which is electrically inert. Injured and ischemic areas on the other hand represent zones that have suffered varying degrees of damage but are still electrically active and can only be recognized by the abnormal electrical effects which they produce. The size of an infarct may be determined by gross and microscopic examination of the heart whereas injury and ischemia, in the sense in which these terms are used by Bayley, cause no recognizable structural changes.

With these ideas in mind it is possible to understand why a precordial electrocardiogram from a patient with a purely subendocardial infarct may show elevation of the RS-T segment and terminal inversion of the T waves. The infarct may not be transmural but the zones of injury and ischemia surrounding it are very likely to extend to the epicardial surface of the heart.

It appears then that electrical effects due to the injury or ischemia associated with infarction are apt to be found in tracings taken by suitably placed indirect leads whether the infarct is subendocardial or transmural. These may be indistinguishable from purely local injury produced in the superficial layers of the epicardium by pericarditis, trauma, or a wide variety of chemical or physical agents. In the latter instances changes in the QRS complex of the type produced by infarction are, of course, absent.

The type of RS-T segment and T wave change seen in the limb leads will depend on the location of the injured and ischemic muscle and the position of the heart with respect to the regions where the extremities join the trunk. The alterations in the standard leads following infarction are usually of the discordant type and may be logically explained by accompanying changes found in unipolar extremity leads. Injury due to pericarditis is likely to produce initial elevation of the RS-T segment in all standard leads usually followed by T wave inversion. Here we know that only the superficial layers of the ventricular wall are responsible for the changes observed and the potentials of the left arm and leg are modified in a similar fashion while those of the right arm are modified in an inverse manner.

During anginal attacks, on the other hand, the transient RS-T displacements are usually downward in all standard leads suggesting predominant injury in the subendocardial layers. There is considerable clinical and experimental evidence to support the view that injury to the subendocardial surface of the ventricle will produce elevation of the RS-T segment in Vr, the unipolar lead from the right upper extremity, (and hence probably in tracings obtained with an electrode in the ventricular cavity) and depression of this segment in the usual precordial and limb leads.[5,6] The transient nature of the injury and ischemia occurring with anginal attacks makes some of the clinical observations difficult to evaluate but experimental studies carried out recently in this laboratory by Pruitt and Valencia[7] provide additional evidence favoring the concept mentioned above.

Interesting observations relating to modifications in the RS-T and T waves have recently been published by Alzamora Castro.[8] He has found that inverted T waves, presumably due to ischemia, occurring in tracings from patients with angina pectoris or recent myocardial infarction may, as a result of exercise, temporarily disappear and be replaced by erect T waves. The explanation for this unexpected and paradoxical finding is not clear but Alzamora Castro points out that, under certain circumstances, upright normal T waves may indicate an increase in the amount of ischemic muscle and reversion to inverted T waves represent an actual improvement in the circulatory state of the myocardium as a whole. In support of this interpretation Alzamora Castro calls attention to more marked inversion of T waves occuring in complexes immediately following a long diastolic pause in tracings taken from similar patients who had auricular fibrillation or premature beats.

It should be clear from the above brief discussion that our knowledge is incomplete regarding many of the phenomena concerned with injury to the myocardium whether it be caused by coronary artery disease or by other factors. Further studies in this field, including use of the catheterization technique now available to obtain intracardiac leads in man, may help to clear up many points which are now in doubt.

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